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VASCULAR DISEASE

REMOTE ISCHEMIC PRECONDITIONING IS NOT MEDIATED BY ENDOGENOUS BRADYKININ IN HUMANS

ACC Oral Contributions
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Background Ischemia reperfusion injury (IRI) induces endothelial dysfunction with attenuation of acetylcholine-induced vasodilatation in man. Remote ischemic preconditioning (RIPC) preserves endothelium-dependent vasodilatation following IRI. Given that exogenous bradykinin protects against IRI, we hypothesised that endogenous bradykinin mediates the vascular protective effects of RIPC.

Methods In a double blind crossover study, 20 subjects were randomised to receive an intravenous infusion of the bradykinin B2 receptor antagonist, HOE-140 (0.1 mg/kg body weight) or saline placebo. IRI (20 min upper arm cuff inflation to 200 mmHg) was induced in the non-dominant arm in all subjects. Prior to induction of IRI 10 subjects also received 3 cycles of 5 min RIPC in the dominant arm. Using bilateral forearm venous occlusion plethysmography, blood flow was measured during intra-brachial infusion of acetylcholine (ACh; 5-20 µg/min) at baseline and following IRI.

Results Compared to baseline, acetylcholine-induced vasodilatation was reduced at 15 and 45 min following IRI, both in the presence (P=0.0002) and absence (P=0.04) of HOE-140. RIPC prevented the reduction in ACh-induced vasodilatation associated with IRI, irrespective of the presence or absence of HOE-140 (P=NS for both).

Conclusions Endothelial vasomotor dysfunction is induced by IRI and can be prevented by RIPC. Our findings suggest that endogenous bradykinin does not play a major role in the induction of IRI or its prevention by RIPC.